AFW
1623

PATENT
Customer No. 22,852
Attorney Docket No. 2405.0167

APPEAL TO THE BOARD OF PATENT APPEALS AND INTERFERENCES

In re Application of:

Mads Liengaard Vigh et al.

Application No.: 09/255,655

Filed: February 23, 1999

For: USE OF D-TAGATOSE AS A PREBIOTIC FOOD COMPONENT

Group Art Unit: 1623

Examiner: H. Owens, Jr.

Commissioner for Patents and Trademarks
Washington, DC 20231

Sir:

TRANSMITTAL OF APPEAL BRIEF (37 C.F.R. 1.192)

Transmitted herewith in triplicate is the APPEAL BRIEF in this application with respect to the Notice of Appeal filed on September 5, 2001.

This application is on behalf of

☐ Small Entity ☒ Large Entity

Pursuant to 37 C.F.R. 1.17(f), the fee for filing the Appeal Brief is:

☐ $160.00 (Small Entity)

☒ $320.00 (Large Entity)

TOTAL FEE DUE:

Notice of Appeal Fee	$320.00
Extension Fee (if any)	$400.00
Total Fee Due	$720.00

☒ Enclosed is a check for $720.00 to cover the above fees.

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PETITION FOR EXTENSION. If any extension of time is necessary for the filing of this Appeal Brief, and such extension has not otherwise been requested, such an extension is hereby requested, and the Commissioner is authorized to charge necessary fees for such an extension to our Deposit Account No. 06-0916. A duplicate copy of this paper is enclosed for use in charging the deposit account.

FINNEGAN, HENDERSON, FARABOW,
GARRETT & DUNNER, L.L.P.

Dated: January 7, 2002

By: Charles E Van Horn
Charles E. Van Horn
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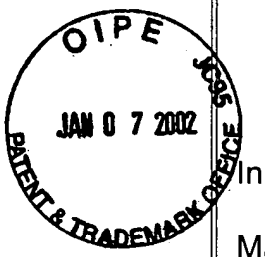

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of:	)	
	)	
Mads Liendgaard Vigh et al.	)	Group Art Unit: 1623
	)	
Application No.: 09/255,655	)	Examiner: H. Owens, Jr.
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	)	
For: USE OF D-TAGATOSE AS A PREBIOTIC FOOD COMPONENT	)	

Assistant Commissioner for Patents
Washington, DC 20231

Sir:

APPEAL BRIEF

In accordance with 37 C.F.R. § 1.192, Appellants submit this Appeal Brief in triplicate with appropriate fee ($320.00), accompanied by a request for a two-month extension of time and a fee of $400.00. Accordingly, submission of the Appeal Brief on or before January 7, 2002 (January 5, 2002 was a Saturday) is timely.

I. Real Party In Interest

The real party in interest in this application is the record owner, Arla Foods AMBA. The assignment was recorded on August 22, 2001 at Reel 12093, starting at Frame 0434.

II. Related Appeals and Interferences

None.

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III. Status of Claims

Claims 1-12 are pending in this application. Claims 1-12 stand rejected under 35 U.S.C. § 103(a).

IV. Status of Amendments

No Amendment was filed subsequent to the date of the Final Office Action (June 6, 2001).

V. Summary of Invention

The claimed invention is directed to either a method for selectively inducing production of butyrate by bacteria in a human colon (claims 1-6) or a method for selectively stimulating growth of lactobacilli and lactic acid bacteria in the human colon (claims 7-12). See Specification at page 6, line 34 to page 7, line 7. The methods include the step of administering D-tagatose, a well-known keto-hexose, in an amount effective to either selectively induce production of butyrate (claims 1-6; Figure 3; page 12, lines 1-6 of the Specification) or selectively stimulate the growth of lactobacilli and lactic bacteria in the human colon (claims 7-12; Figure 5; page 16, lines 6-11 of the Specification).

VI. Issues

Whether claims 1-12 are unpatentable under 35 U.S.C. § 103(a) over the teachings of Zehner (U.S. Patent No. 4,786,722) in combination with Morelli et al. (U.S. Patent No. 5,709,857), MacFarlane et al., "The Calonic Flora, Fermentation, and Large Bowel Digestive Function," *The Large Intestine*: Physiology, Pathophysiology and Disease, Chapter 4, pages 51-92 (1991), and Mortensen et al., "Short-Chain Fatty Acid Production from Mono-and Disaccharides in a Fecal Incubation System: Implications

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for Caloric Fermentation of Dietary Fiber in Humans," *American Institute of Nutrition*, pages 321-325 (1987).

VII. Group of Claims

For purposes of this appeal, claims 1-6 stand or fall together separate from claims 7-12 that stand or fall together.

VIII. Arguments

Claims 1-12 stand rejected under 35 U.S.C. § 103 as being unpatentable over Zehner (U.S. Patent No. 4,786,722) in combination with Morelli (U.S. Patent No. 5,709,857), MacFarlane (*The Large Intestine*: Physiology, Pathophysiology and Disease (1991)) and Mortensen et al., (*American Institute of Nutrition* (1988)). This rejection, as set forth in the Final Office Action of June 6, 2001, fails to establish a *prima facie* case of obviousness of the claimed invention because: (1) the Office has not properly addressed the "selectively" limitation in all the claims on appeal; (2) the prior art relied upon fails, either individually or collectively, to establish a motivation to administer D-tagatose in an amount effective to produce the claimed results; (3) the prior art relied upon fails, either individually or collectively, to establish a reasonable predictability that the administration of D-tagatose would produce the effects recited in the claims on appeal; and (4) the prior art relied upon fails, either individually or collectively, to teach all limitations of the claims on appeal. After discussing the prior art relied upon in the rejection, the reasons that the Office has failed to establish a *prima facie* case of obviousness will be discussed.

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PRIOR ART

Zehner teaches that D-tagatose, a naturally-occurring keto-hexose, can be used as a sweetener or bulking agent to prepare a sweetened edible formulation. (Column 1, lines 40-56). Tests with D-tagatose have shown that it is not absorbed across the intestinal membrane and that it is not degraded significantly in the small intestine or in the lower intestine by indigenous micro flora. (Column 2, lines 20-36). It was also reported that D-tagatose was fermented by certain human micro flora such as *lactobacillus casei*, and that some degradation of D-tagatose in the human colon can be expected. Zehner teaches (col. 3, lines 4-32) that D-tagatose may be added to a large variety of edible materials or foodstuffs in an amount to attain the desired level of sweetness. There is nothing in Zehner to teach or suggest that the expectation of some degradation of D-tagatose in the human colon would be associated with either the production of butyrate or the growth of lactobacilli and lactic acid bacteria in the human colon, to say nothing of the selective production of butyrate or the selective stimulation of the growth of lactobacilli and lactic acid bacteria in the human colon.

MacFarlane et al. describes the complex of microbial interactions that occur in the human gastrointestinal tract. This article notes (page 55) that the human gut micro flora is in intimate contact with its host and participates in a vast number of metabolic activities, the products of which can interact with the host in a variety of ways. It is noted (page 56, column 1)) that the majority of carbohydrate fermenting species in the colon produce short chain fatty acids (SCFA) acetate, propionate, and butyrate, and the gases carbon dioxide and hydrogen as the principal end products. It is further noted (page 56, column 2) that the products of fermentation are determined by the amount

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and type of substrate, the rate and extent to which it is broken down, the type of flora involved, and host factors such as transit time. Although MacFarlane et al. does identify lactobacillus as a known type of human gut micro flora (p. 54, column 2), MacFarlane et al. does not contain a specific teaching relied on by the Examiner on page 2 of the Final Office Action that "bacterial populations such as lactobacilli grow on carbon sources supplied by substrates such as monosaccharides and disaccharides (pages 52-56)." MacFarlane et al. contains no description of D-tagatose nor the role it or any other sugar may have in selectively inducing the production of butyrate or in stimulating the growth of lactobacilli and lactic acid bacteria in the human colon, to say nothing of the selective stimulation of the growth of lactobacilli and lactic acid bacteria in the human colon.

Mortensen et al. describes the results of *in vitro* faecal incubation system tests designed to demonstrate how certain compounds influence short chain fatty acid (SCFA) production in the colon. An *in vitro* system was used because these biochemical mechanisms are difficult to assess *in vivo*. These tests were performed with certain hexoses, pentoses, and uronic acid monomers along with other mono- and disaccharides (page 322, column 1). The study suggests (page 324) that a substantial capacity for enhancement of SCFA production is available when sufficient amounts of an appropriate substrate are present, and that the proportions between the individual SCFAs produced may be substrate dependent (The Abstract characterizes this suggestion as speculation). Mortensen et al. does not describe D-tagatose, nor is there any description of the ability of D-tagatose to selectively induce the production of butyrate or to stimulate the growth of lactobacilli and lactic acid bacteria in the human

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colon, to say nothing of the selective stimulation of the growth of lactobacilli and lactic acid bacteria in the human colon.

Morelli et al. teaches that several species of lactobacillus isolated from the human colon are able to degrade a large variety of carbohydrates including well-known and commercial malabsorbed carbohydrates like mannitol and maltose. D-tagatose is also identified in charts 1-4 as providing a positive indication for carbohydrate fermentation. The mere fact, however, that these isolated species can degrade sugars such as D-tagatose, does not mean that they would promote the growth of lactobacilli or lactic acid bacteria in the competitive environment of the colon. Growth on various sugars is an old and well recognized tool to characterize isolated bacteria and discriminate between genera and species. However, these published data do not give any idea on how well bacteria grow on the substrates. In order for a carbohydrate to promote growth of lactobacilli in the colon, it has to be selectively degraded by lactobacilli in the highly competitive environment of the colon. This is particularly unpredictable because most other bacteria and numerous colon bacteria have, similar to lactobacillus, a rather broad ability to degrade malabsorbed carbohydrates like fibers, polyols and sugars and thus prevent selective growth of beneficial bacteria like lactobacillus by simple competition. Although Morelli et al. may confirm that D-tagatose can be used as a tool to characterize isolated bacteria, there is no suggestion that it can be used in the manner recited in the instant claims to achieve the claimed results.

DIFFERENCES BETWEEN THE PRIOR ART AND THE CLAIMED INVENTION

Although the differences between the claimed invention and each of the individual references relied upon by the Examiner in the Final Rejection have been

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identified above, the principal difference between the prior art and the claimed invention is the failure of the prior art to suggest or predict that D-tagatose will selectively induce the production of butyrate in the colon and selectively stimulate the growth of beneficial lactobacilli and lactic acid bacteria in the human colon at levels that are not contemplated or expected from the teachings of the prior art. The Office, in providing an explanation of the rejection in the Final Office Action, consistently ignores the "selective" limitation in all the claims on appeal. Thus, for example, the Final Office Action at page 3 states:

> Mortensen et al. and MacFarlane et al. teach the production of butyrate from monosaccharide and disaccharide substrates broadly in the human colon, which differs from the instantly claimed invention only with respect to the fact that D-tagatose is not specifically cited;

Although Mortensen et al. and MacFarlane et al. teach that the production of butyrate could be expected from some monosaccharide or disaccharide substrates, the absence of a specific description of D-tagatose is not the only difference between the teachings of those references and the claimed invention. All claims on appeal clearly require that D-tagatose be administered to a human in an amount to selectively achieve certain results. This limitation is also a difference between the claimed invention and the references relied upon in the Final Office Action.

The Office Action further argues at page 4 of the Final Office Action:

> Moreover, one of skill in the art would have a reasonable expectation of success for the production of butyrate or the growth of Lactobacilli with not only D-tagatose, but any other mono- or disaccharide which escapes digestion in the small intestine and has been indicated by the prior art as a substrate for commensalistic flora from which SCFA's such as butyrate are produced.

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Again, the Examiner has failed to take into account the limitations of the claimed invention particularly with respect to the requirement that D-tagatose be administered in an amount that is effective to achieve certain selective results that are not predictable from the prior art. Not only does the prior art fail to provide any reasonable expectation of success of achieving these selective results by administering D-tagatose, Mortensen et al. clearly shows that the results achieved regarding the production of butyrate are NOT predictable.

PRIOR ART FAILS TO PROVIDE
MOTIVATION OR SUGGESTION TO USE D-TAGATOSE

The Office carries the initial burden of establishing a *prima facie* case of obviousness. In doing so, it must establish: (1) that there exists some suggestion or motivation, either in the references themselves or in the knowledge generally available to one of ordinary skill in the art, to combine reference teachings; (2) that there is a reasonable expectation of success that can be derived by such a combination; and (3) that the references when combined teach or suggest all the limitations of the claims. As to the first element, the Federal Circuit has stated that the evidence of the teaching, suggestion, or motivation to combine references must be "clear and particular." *In re Dembiczak*, 50 USPQ2d 1614, 1617 (Fed. Cir. 1999). The prior art relied upon by the Examiner simply does not provide a clear and particular suggestion to administer D-tagatose to selectively induce the production of butyrate or selectively stimulate the growth of lactobacilli and lactic acid bacteria in the colon.

Neither Mortensen et al. nor MacFarlane et al., alone or in combination, teach or suggest that the administration of D-tagatose will produce the selective effects recited in these claims. As noted above, these references show that it is generally

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recognized that some monosaccharides and some keto-hextoses may serve as a substrate for the production of short chain fatty acids, and have been associated with fermentation by certain bacteria. However, these teachings do not remotely resemble a clear and particular motivation to use D-tagatose to produce the selective effects recited in the claims on appeal.

The teachings of Mortensen et al. could probably be argued as being closest to the claimed invention in that it speculates that the production of some SCFA can be enhanced depending on the substrate used. However, of the fifteen compounds specifically used in the tests reported in Mortensen et al., only four showed an increase in butyrate production. While L-glucose showed a slight increase in butyrate production, the other seven monosaccharides tested (D-gelactose, D-fructose, D-glucose, D-arabinose, D-xylose, D-ribose and D-mannose) showed essentially no increased amount of butyrate. The three other compounds with high production of butyrate were reported as sorbitol (a sugar alcohol) and the two aldonic acids (uronic acid monomers), D-galacetoronic acid and D-glucuronic acid. The other polyol (D-mannitol) and the disaccharides (lactose and lactulose) tested showed essentially no increased amount of butyrate. Clearly these test results do not point in any particular direction or provide any motivation for selecting any particular compound, and specifically D-tagatose, to obtain enhanced butyrate production. The teachings of Morelli et al. and Zehner are silent with respect to any motivation to associate the administration of D-tagatose with butyrate production in any amount.

Similar to the arguments presented above with respect to claims 1-6 and the administration of D-tagatose to selectively induce production of butyrate, the

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references relied upon by the Office fail to provide any motivation or suggestion to administer D-tagatose in an amount effective to selectively stimulate the growth of lactobacilli and lactic acid bacteria in the human colon. Mortensen et al. and MacFarlane et al. contain little or no discussion or description of the interaction of any sugar or monosaccharide with any specific bacteria, and certainly nothing about D-tagatose. While Morelli et al. describes that certain lactobacillus species isolated from the human colon are able to degrade carbohydrates such as D-tagatose, there is no indication or suggestion that it would promote the growth of lactobacilli and lactic acid bacteria in the competitive environment of the human colon. Although Zehner does suggest that some degradation of D-tagatose could be expected to occur in the colon, there is no suggestion that this degradation would be associated with the selective stimulation of the growth of lactobacilli and lactic acid bacteria in the human colon when administered in an effective amount.

It appears to be the position of the Office that since it is known that certain effects can occur when certain sugars are administered to a human, it would be obvious to select any sugar to see what effects would occur. However, this "obvious to try" approach to establish a *prima facie* case of obviousness is legally incorrect, particularly when it's coupled (as here) with an utter lack of predictability of achieving the recited selective effects. *In re Yates*, 211 USPQ 1149, 1151 (C.C.P.A. 1981).

PRIOR ART FAILS TO ESTABLISH A REASONABLE EXPECTATION OF SUCCESS WHEN ADMINISTERING D-TAGATOSE

The prior art relied upon in the Final Office Action fails to provide a reasonable expectation that the administration of D-tagatose in an effective amount would produce the selective effects recited in all claims on appeal. Rather than

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providing a reasonable expectation of success, the prior art relied on by the Office clearly demonstrates the unpredictable nature of the effects produced by administering a sugar to a human. Specifically, Mortensen et al. not only fails to address the effects of D-tagatose, but clearly shows (Figures 1 and 2), that there is a lack of predictability of the amounts of various SCFAs that are produced even *in vitro* by several mono- and disaccharides and other compounds. Further, MacFarlane et al. teaches (page 61, column 1) that intestinal bacteria degrade different polysaccharides at different rates, and that (page 63, column 2) one problem with studies of SCFA production and metabolism is that each fatty acid is metabolized differently and at a number of sites in the body. Carbohydrate fermentation in the human colon is difficult to study *in vivo*, and the comparison of the results of *in vitro* experiments with the *in vivo* human data was described as difficult at column 2, page 216 of Edwards et al. (In Vitro Method for Quantification of the Fermentation of Starch by Human Faecal Bacteria, *J. Sci. Food Agric.* 71:209-217 (1996) a document of record and discussed in the reply filed March 1, 2000). Clearly, these documents fail to provide any basis for establishing a predictability of success; in fact, they clearly demonstrate the unpredictable nature of trying to select an appropriate substrate to achieve the claimed effects.

Morelli et al. and Zehner provide no disclosure or basis to predict what SCFAs, if any, would be produced. Accordingly, there can be no reasonable predictability gleaned from the prior art relied upon in the Final Office Action that an administration of an effective amount of D-tagatose would selectively induce production of butyrate as recited in claims 1-6 in this application.

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Further, the teachings of Mortensen et al. and MacFarlane et al. do not provide any basis for predicting the results required by the method of claims 7-12. As recognized by the Examiner, Morelli et al. shows that some lactobacillus species isolated from the human colon are able to degrade a large variety of carbohydrates. This observation, however, does not mean, nor is it predictable, that these carbohydrates would selectively stimulate growth of lactobacilli and lactic acid bacteria in the competitive environment of the human colon. Likewise, although Zehner does suggest that it is likely that D-tagatose would undergo some degradation in the colon, there is no suggestion or teaching that would suggest that this degradation would be associated with the selective stimulation of the growth of lactobacilli and lactic acid bacteria in the human colon. Accordingly, the prior art relied upon by the Office fails to provide any information that would permit a person of ordinary skill in the art to predict that success would be achieved in selectively stimulating the growth of these bacteria upon administration of D-tagatose in an effective amount.

PRIOR ART FAILS TO TEACH ALL THE LIMITATIONS OF THE CLAIMS ON APPEAL

The Office has failed to establish that the prior art relied upon in the Final Office Action teaches all of the limitations of these claims. Specifically, the Office has failed to establish that the administration of D-tagatose in an effective amount will "selectively" induce butyrate production (claims 1-6) or "selectively" stimulate the growth of lactobacilli and lactic acid bacteria in the human colon (claims 7-12). The Office has addressed these limitations in ways that are factually and legally insufficient to establish that the prior art teaches or renders obvious these limitations.

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Specifically, on page 5 of the Final Office Action, the Office has stated that:

> . . . with regards to the "selective" production of butyrate, the prior art (Mortensen et al. and MacFarlane et al.) has set forth that monosaharrides or keto-hexoses serve as a substrate for the production of Short Chain Fatty Acids such as butyrate and also allow for the growth of commensalistic indigenous flora such as lactobacilli.

The Office has relied on what are at best very general teachings to provide support for the conclusion that the prior art teaches that D-tagatose will "selectively" induce butyrate production and "selectively" stimulate the growth of lactobacilli and lactic acid bacteria in the human colon. Even though the teachings of Mortensen et al. and MacFarlane et al. are general in nature, the Office has over-generalized the teachings. While it is true that these references teach that some monosaharrides and some keto-hexoses may serve as a substrate for the production of some short chain fatty acids, there is clearly no suggestion or predictability that all monosaharrides or keto-hexoses will serve as a substrate for the production of all short chain fatty acids, or the selective production of butyrate. In fact, the teachings of Mortensen et al. clearly demonstrate that the enhanced production of any particular short chain fatty acid by the compounds tested is unpredictable. How can these teachings possibly suggest the use of D-tagatose? Further, there is nothing in either Mortensen et al. or MacFarlane et al. that would suggest or provide the basis for predicting that any particular compound would stimulate the growth of lactobacilli and lactic bacteria in the competitive environment of the human colon.

The Office further observed (page 5 of the Final Office Action) that:

> Mortensen et al. teaches that a substantial capacity for enhancement of the short chain fatty acid (propionate and

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> butyrate specifically) production is available when sufficient amounts of an appropriate substrate are present (p. 324, paragraphs 2); therefore, D-tagatose is not alone in the production of butyrate in the human colon;

These observations also are legally insufficient to establish that the administration of an effective amount of D-tagatose will "selectively" induce butyrate production and "selectively" stimulate the growth of lactobacilli and lactic acid bacteria in the human colon. There is no suggestion or direction in Mortensen et al., or any of the prior art relied on, as to how one would select an appropriate substrate for the selective inducement of butyrate production. The fact that D-tagatose is not alone in the production of butyrate in the human colon is irrelevant to the question of obviousness in this application. The question is not whether D-tagatose is the only sugar to possess this capability, but whether there is anything in the prior art that would suggest or permit one skilled in the art to predict that D-tagatose would produce the effects recited in these claims. As to the latter question, the answer is NO.

Finally, the Office observed on page 5 of the Final Office Action that:

> Zehner clearly teaches that the state of the art has recognized the fermentation of D-tagatose by human microflora, specifically *Lactobacillus casei* (column 2, lines 56-67). Zehner also teaches that this fermentation could be beneficial if it is slow in the human gut and produces non-caloric metabolites (column 2, line 67-column 3, line 3).

Again, the observation that Zehner teaches that D-tagatose may be expected to be degraded by some human micro flora, or that this fermentation could be beneficial under certain conditions that have not been shown to exist, fails in any way to suggest or permit one skilled in this art to predict that the administration of an effective amount of D-tagatose would provide the selective results required by all claims in this application. There is no teaching in Zehner that would associate the degradation of

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D-tagatose with a selective stimulation in the growth of lactobacilli and lactic acid bacteria. Further, the broad speculative suggestion that benefits might be obtained if certain conditions exist cannot be deemed a clear and particular teaching to use D-tagatose to achieve any specific result.

For the reasons discussed above, the Office has failed to establish any of the essential elements of a *prima facie* case of obviousness based on the references relied upon in the Final Office Action. The Board is respectfully requested to reverse the rejection of claims 1-12 under 35 U.S.C. § 103 to put this application in condition for allowance.

IX. Appendix

An Appendix is attached containing a copy of claims 1-12 involved in this appeal.

Please grant any extensions of time required to enter this Appeal Brief and charge any additional required fees to our Deposit Account No. 06-0916.

Respectfully submitted,

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Dated: January 7, 2002

By: Charles E Van Horn
Charles E. Van Horn
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Appendix

1. A method for selectively inducing production of butyrate by bacteria in the human colon comprising administering D-tagatose to a human in an amount effective to selectively induce production of butyrate.

2. A method according to claim 1 wherein D-tagatose is administered orally.

3. A method according to claim 2 where D-tagatose is administered in a daily amount of 5 to 30 grams.

4. A method according to claim 3 wherein the daily amount is 5 to 15 grams.

5. A method according to claim 2 wherein D-tagatose is administered orally in a food product.

6. A method according to claim 5 wherein the food product is selected from the group consisting of a confectionery, chewing gum, ice cream, dessert, soft drink, breakfast cereal, yogurt, health drink and health bar.

7. A method for selectively stimulating growth of lactobacilli and lactic acid bacteria in the human colon comprising administering D-tagatose to a human in an amount effective to selectively stimulate growth of lactobacilli and lactic bacteria in the human colon.

8. A method according to claim 7 wherein D-tagatose is administered orally.

9. A method according to claim 8 wherein D-tagatose is administered in a daily amount of 5 to 30 grams.

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10. A method according to claim 9 wherein the daily amount is 5 to 15 grams.

11. A method according to claim 8 wherein D-tagatose is administered orally in a food product.

12. A method according to claim 11 wherein the food product is selected from the group consisting of a confectionery, chewing gum, ice cream, dessert, soft drink, breakfast cereal, yogurt, health drink and health bar.

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